Amendments to the Specification:

Please replace the paragraph beginning at page 3, line 16, with the following amended paragraph:

--Growth inhibitory proteins typically cause growth cone collapse, a process that causes dramatic rearrangements to the growth cone cytoskeleton (Bandtlow, C.E., et al., (1993) *Science* 259, 80-83; Fan, J., *et al.*, (1993) *J. Cell Biol.* 121, 867-878; Li, M., *et al.*, (1996) *J. Neurosci. Res.* 46, 404-414). One family of proteins that has been implicated in receptor-medicated signaling to the cytoskeleton is the small GTPases of the Rho family (Hall, A. (1996) *Ann. Rev. Cell Biol.* 10, 31-54). In non-neuronal cells it has been clearly documented that mutations in Rho family members that include Rho, Rac and cdc42, affect adhesion, actin polymerization, and the formation of lamellipodia and filopodia, which are all processes important to motility (Nobes, C.D. and Hall, A.R. (1995) *Cell* 81, 53-62). There is now good evidence that members of the Rho family regulate axon outgrowth in development. Mutations in Rho-related family members block the extension of axons in Drosophila (Luo, L., et al., (1994) *Genes Dev.* 8, 1787-1802) and disrupt axonal pathfinding in C. elegans (Zipkin, I.L., et al., (1997) *Cell* 90, 883-894). More recently it has been shown that the guidance molecule collapsin acts through a Rac-dependent mechanism (Jin, Z. and Strittmatter, S.M. (1997) *J. Neurosci.* 17, 6256-6263). In transgenic mice that express constitutively active Rac in Purkinje cells, there are alterations in the development of axon terminals and dendritic arborizations (Luo, L., *et al.*, (1996) *Nature* 379, 837-840). Consistent with the observations *in vivo*, it was found that dominant negative Rac expressed in PC12 cells disrupts neurite outgrowth in response to NGF ~~(Hutchens, J.A., *et al.*, (1997) *Molec. Biol. Cell* 8, 481-500)~~ (Lamoureux *et al.* (1997) *J. Cell. Sci.* 110(Pt2), 635-641). Also, treatment of PC12 cells with lysophosphatidic acid, a mitogenic phospholipid, causes neurite0 retraction that is mediated by Rho (Tigyi, G., *et al.*, (1996) *J. Neurochem.* 66, 537-548). Therefore, different members of the Rho family can exert distinct effects on neurite growth, and in PC12 cells the activation of Rho is correlated with growth cone collapse. In non-neuronal cells, Rho participates in integrin-dependent signaling (Laudanna, C., *et al.*, (1996)

Science 271, 981-983; Udagawa, T. and McIntyre, B.W. (1996) *J. Biol. Chem.* 271, 12542-12548). The possibility that Rho might play a role within the myelin-derived growth inhibitory system has been studied (Jin, Z. and Strittmatter, S.M. (1997) *J. Neurosci.* 17, 6256-6263). It was concluded, however, that the inhibitory effects of myelin are not mediated by Rho family members.--